

POSITION STATEMENT

Fruit, vegetables and cancer prevention



Key messages

Fruit and vegetables are high in nutrients that are potentially protective against cancer. They also play an important role in weight management. As obesity is a *convincing* risk factor for cancer of the colorectum, kidney, pancreas, oesophagus, endometrium and breast (in post-menopausal women), fruit and vegetables may also protect against cancer indirectly by helping to maintain a healthy body weight.

Although there has been a slight weakening of the evidence supporting the role of fruit and vegetables in reducing the risk of some cancers, overall the evidence is suggestive of a protective effect. Fruit and vegetables appear to protect against cancers of the digestive tract, such as cancer of the mouth, pharynx, larynx, oesophagus, stomach and colorectum. Fruit may also protect against lung cancer.

There does not appear to be an association between fruit and vegetable consumption and prostate or breast cancer. However, foods containing lycopene such as tomato may reduce the risk of prostate cancer.

Cancer Council supports the Australian Dietary Guidelines that recommend eating plenty of fruit and vegetables, and the population recommendation of at least **two serves of fruit** and **five serves of vegetables** daily.

Because the knowledge is still incomplete about the ways in which nutrients may reduce cancer risk, Cancer Council also recommends that people eat a **variety** of different fruit and vegetables to obtain maximum benefits. Fruit and vegetables are best consumed fresh and whole (i.e. not in a supplement form) and consumption of both cooked and raw vegetables are recommended.

For people already diagnosed with cancer, there is some evidence that a diet high in fruit and vegetables is not of significant benefit, but is unlikely to be harmful. Cancer Council recommends the general community guidelines of two serves of fruit and five serves of vegetables daily for cancer survivors.

Background

The protective effects of fruit and vegetables against cancers, as well as other diseases such as coronary heart disease and type 2 diabetes, has led to the promotion of fruit and vegetables consumption as a national public health priority.¹

Fruit and vegetables are high in nutrients such as fibre, vitamins, minerals, antioxidants and phytochemicals, which are chemicals found in plants such as flavonoids, carotenoids and lignans. It is probably a combination of these nutrients and phytochemicals found together in whole foods that helps to reduce the risk of chronic diseases rather than one anti-cancer component, although many different mechanisms have been proposed.² Single nutrients identified from the analysis of epidemiological studies have usually been unsuccessful when investigated further in trials, making the whole food approach more appropriate to prevention advice.

Fruit and vegetables also play an important role in weight management due to their low energy density, high fibre content and capacity to displace higher energy foods from the diet. Obesity is a known risk factor for cancer of the colorectum, kidney, pancreas, oesophagus, endometrium and breast (in post-menopausal women).³⁻⁵ Evidence suggests that obesity may also be linked to cancer of the gallbladder and liver.⁴ Therefore fruit and vegetables may reduce the risk of cancer directly through the provision of specific anti-carcinogenic agents and indirectly through their role in weight management.

Rationale

The International Agency for Research on Cancer (IARC) concluded that 5-12% of cancers could be attributed to low fruit and vegetable consumption.⁶ Australian data suggests that 2% of cancers were attributable to low consumption of fruit and vegetables.⁷ In terms of health care costs, it has been estimated that low vegetable intake (<4 serves per day) accounts for 17% of the cost of bowel cancer, 2% of the cost for breast cancer, and 9% of the cost of lung and of prostate cancer.⁸ Twenty one percent of the cost of lung cancer and 4% of the cost of breast cancer has been attributed to lower fruit intake (<3 serves per day).⁸

Many cancer associations worldwide, together with a number of National Dietary Guideline committees have recommended a daily intake of 5-7 serves of fruits and vegetables to reduce cancer risk. Therefore it is important for the Cancer Council to evaluate the evidence on fruit, vegetables and cancer prevention in order to develop its messages and recommendations.

This position statement summarises epidemiological evidence from major cancer prevention reports, meta-analyses and systematic reviews that examined the relationship between fruit and vegetable consumption and cancer risk. In addition to the meta-analyses and systematic reviews identified, several cohort studies were also considered, including the published results of the European Prospective Investigation into Cancer and Nutrition (EPIC) study. A major strength of EPIC is the wide range of fruit and vegetable intakes that have been assessed.

Views on fruit and vegetables in cancer prevention reports

Several major reports that have investigated the relationship between fruit and vegetable intake and cancer risk show that there has been a shift in the strength of the evidence over the last 10 years. These reports indicate that the evidence appears to be somewhat weaker than previously thought (Table 1). However despite this, fruit and vegetable consumption continues to play an important role in cancer prevention. On the whole, evidence that vegetables are protective is stronger than for fruits, but this may simply reflect the generally greater consumption of vegetables worldwide or the different mix of nutrients obtained from them.

Table 1. Conclusions from the major cancer prevention reports regarding the cancer protective effect of fruit (f) and vegetables (v).

Organisation Review	Highest Evidence Convincing	Moderate Evidence Probable	Lower Evidence Possible / Limited
WCRF/AICR (2007) ⁴		Mouth (f&v) Pharynx (f&v) Larynx (f&v) Oesophagus (f&v) Stomach (f&v) Lung (f)	Nasopharynx (f&v) Colon & rectum (f&v) Pancreas (f) Liver (f) Lung (v) Ovary (v) Endometrium (v)
IARC (2003) ⁶		Oesophagus (f&v) Stomach (f) Colon & rectum (v)	Mouth (f&v) Pharynx (f&v) Larynx (f&v) Kidney (f&v) Colon & rectum (f) Bladder (f) Stomach (v) Lung (v) Ovary (v)
WHO/FAO (2003) ⁵		Oral Cavity (f&v) Oesophagus (f&v) Stomach (f&v) Colon & rectum (f&v)	
COMA (1998) ⁹	Oesophagus (f&v)	Stomach (f&v) Colon & rectum (v)	Breast (f&v)
WCRF/AICR (1997) ¹⁰	Mouth (f&v) Pharynx (f&v) Oesophagus (f&v) Stomach (f&v) Colon & rectum (v) Lung (f&v)	Larynx (f&v) Pancreas (f&v) Breast (f&v) Bladder (f&v)	Ovaries (f&v) Cervix (f&v) Endometrium (f&v) Thyroid (f&v) Liver (v) Prostate (v) Kidney (v)

In 2007, the World Cancer Research Fund and the American Institute of Cancer Research (WCRF/AICR) published an update of their conclusions regarding fruit and vegetables in their *Report on Food, Nutrition Physical Activity and the Prevention of Cancer*.⁴ The report found that there was *probable* evidence of a protective effect by fruit and vegetables for cancer of the mouth, pharynx, larynx, oesophagus and stomach, and fruit also *probably* protected against lung cancer (Table 1).⁴ There was *limited suggestive* evidence that fruit and vegetables reduced the risk of nasopharyngeal and colorectal cancer, fruit lowered the risk of pancreatic and liver cancer and vegetables reduced the risk of lung, ovarian and endometrial cancer.⁴

For particular types of fruits and vegetables, WCRF found that allium vegetables *probably* protected against stomach cancer, garlic *probably* protected against colorectal cancer and foods containing lycopene *probably* protected against prostate cancer.⁴ There was *limited suggestive* evidence that carrots lowered the risk of cancer of the cervix.⁴

The IARC reviewed the evidence relating to cruciferous vegetables, isothiocyanates and indoles in 2004 and found that for human studies:¹¹

- There was limited evidence that cruciferous vegetables reduced the risk of stomach and lung cancer.
- There was inadequate evidence that cruciferous vegetables reduced the risk for cancers at all other sites.
- There was inadequate evidence to assess the independent effects of isothiocyanates and indoles on human cancer risk, as opposed to their combined effects with other compounds in cruciferous vegetables.

In 2003, IARC found that a high intake of vegetables *probably* reduced the risk of oesophageal and colorectal cancer, while a high intake of fruit *probably* reduced the risk of oesophageal and stomach cancer (Table 1).⁶ However the IARC rated the evidence for an association between vegetable intake and cancer of the mouth, pharynx, larynx, kidney, stomach, lung and ovary and the evidence for an association between fruit intake and cancer of the mouth, pharynx, larynx, kidney, colorectum and bladder as *possible*.⁶

Similarly, the World Health Organization (WHO) suggested that a high intake of fruit and vegetables *probably* reduced the risk of cancers of the oral cavity, oesophagus, stomach and colorectum (Table 1).⁵ WHO recommended an intake of at least 400g of fruit and vegetables daily (in addition to potatoes).

The conclusions from WHO and IARC differed from previous reviews by WCRF/AICR¹⁰ and the United Kingdom Department of Health Committee of the Medical Aspects of the Food Supply (COMA)⁹ because of the inclusion and consideration of more prospective studies published since these original reviews were undertaken. In recent years it has been increasingly apparent that the protective association between fruit and vegetable intake and cancer is much stronger in case-control studies than in cohort studies. Furthermore, randomised controlled trials involving fruit and vegetable intake, notably for colorectal cancer, have not demonstrated any benefit. Case-control studies are known to be prone to recall and selection bias, however the length of follow-up in cohort studies can also potentially limit the interpretation of their results.

Results published in 2001 from the EPIC study showed that significant health gains are made from even a small increase in fruit and vegetable intake.¹² Increasing intakes of fruit and vegetables by just 50g a day (equivalent to 2/3 cup cooked vegetables or 1/3 of a piece of fruit) was associated with a reduction in cancer risk of around 20%.¹²

Epidemiological evidence

Recent reviews and meta-analyses suggest that fruit and vegetables may protect against oral, laryngeal, oesophageal, colorectal and lung cancer. Fruit consumption may also lower the risk of stomach and bladder cancer. Fruit and vegetables do not appear to be associated with a lower risk of prostate, breast or ovarian cancer. However, one meta-analysis does suggest that tomato and lycopene intake may reduce the risk of prostate cancer.¹³

All cancers combined

The association between fruit and vegetable intake and the risk of major chronic disease was examined in the Nurses Health Study and Health Professionals Study.¹⁴ There was no association between the consumption of fruit and vegetables and cancer incidence. The relative risk (RR) of cancer based on a continuous measure for the increment of five serves per day of total fruit and vegetable intake was 1.00 (95% confidence interval (CI)= 0.95-1.05).¹⁴ Interestingly, five serves of fruit and vegetables were protective against cardiovascular disease (RR= 0.88, 95% CI= 0.81-0.95).¹⁴

Oral cancer

In 2006, a meta-analysis of 15 case-control studies and one cohort study found a statistically significant reduction in oral cancer risk for each portion of fruit (combined adjusted odds ratio (OR)= 0.51, 95% CI= 0.40-0.65) and vegetable consumed (combined adjusted OR= 0.50, 95% CI= 0.38-0.65).¹⁵ Similar results were found when results were pooled from 12 studies that adjusted for age, sex, smoking and alcohol intake (OR for fruit= 0.49, 95% CI= 0.39-0.63; OR for vegetables= 0.43; 95% CI= 0.31-0.59).¹⁵ Interestingly citrus fruit appeared to provide greater protection than overall fruit consumption (OR= 0.38, 95% CI 0.26-0.56).¹⁵

A meta-analysis completed in 2003 found that in case-control studies (nine studies on fruit and seven studies on vegetable consumption), both fruit (RR= 0.53, 95% CI= 0.37-0.76) and vegetables (RR= 0.84, 95% CI= 0.67-1.07) provided protection against oral cancer, with fruit providing a statistically significant result.¹⁶

Laryngeal cancer

In the only meta-analysis identified for laryngeal cancer, fruit consumption provided a significant protective effect (RR= 0.73, 95% CI= 0.64-0.84), while vegetables appeared to provide a very small non-significant reduction in risk (RR= 0.92, 95% CI= 0.83-1.02) in case-control studies (five studies on fruit and seven studies on vegetable consumption).¹⁶

This meta-analysis was very limited, as out of the eight case-controls studies identified, one was excluded from the analysis on both fruit and vegetables, while two were excluded from the analysis on fruit. The authors point out that the first study was excluded because study subjects were classified into only two categories of consumption, however it is not known why the other two studies were excluded.

Oesophageal cancer

The effect of fruit and vegetable intake on the risk of adenocarcinoma of the oesophagus was investigated as part of the EPIC study.¹⁷ A non-significant inverse association was found for total vegetable consumption (hazard ratio (HR)= 0.71, 95% CI= 0.34-1.48), total fruit intake (HR= 0.94, 95% CI= 0.49-1.80) and citrus fruit consumption (HR= 0.73, 95% CI= 0.39-1.37).¹⁷

An increase of 100g per day of total vegetables (HR= 0.72, 95% CI= 0.32-1.64), total fresh fruit (HR= 0.84, 95% CI= 0.60-1.17) and citrus fruit (HR= 0.77, 95% CI= 0.46-1.28) also showed a non-significant inverse association with oesophageal cancer.¹⁷ A limitation of the EPIC study was that the number of oesophageal cancer cases was small, which limits the statistical power of the study.

A meta-analysis completed in 2003, which included one cohort study and 12 case-control studies, indicated a significant protective association for fruit (RR= 0.72, 95% CI= 0.62-0.83) and vegetables (RR= 0.89, 95% CI= 0.82-0.97) with oesophageal cancer.¹⁶

Stomach cancer

The effect of fruit and vegetable intake on the risk of gastric cancer was investigated as part of the EPIC study.¹⁷ The range of fruit and vegetable intakes varied greatly between the lowest intakes and highest intakes. There was very little evidence for a protective association between fresh fruit (HR= 0.99, 95% CI= 0.68-1.42) and stomach cancer, while there was a non-significant inverse association for citrus fruit (HR= 0.88, 95% CI= 0.63-1.24) and a non-significant positive association for vegetables (HR= 1.15, 95% CI= 0.78-1.70) and stomach cancer.¹⁷

In 2005, a meta-analysis which included 14 cohort studies on the association between stomach cancer and the consumption of fruit (13 studies) and vegetables (eight studies) found a slight non-significant protective effect for fruit (RR= 0.89, 95% CI= 0.78-1.02), while only a very small non-significant inverse relationship was found for vegetable consumption (RR= 0.98, 95% CI= 0.86-1.13).¹⁸ The strongest association was seen in those studies with a follow-up period ≥ 10 years for fruit (RR= 0.66, 95% CI= 0.73-0.93) and vegetables (RR= 0.71, 95% CI= 0.53-0.94).¹⁸ Fruit consumption had a non-significant negative affect on mortality (RR= 0.88, 95% CI= 0.70-1.09), while vegetable consumption had a small non-significant positive affect on mortality (RR= 1.02, 95% CI= 0.78-1.33) in those studies with a follow-up period ≥ 10 years.¹⁸

A meta-analysis from 2003 included seven cohort studies and 24 case-control studies regarding fruit consumption and stomach cancer, and five cohort studies and 17 case-control studies regarding vegetable consumption and stomach cancer.¹⁶ All but one of these cohort studies was included in the meta-analysis mentioned above.¹⁸

The combined analysis of case-control and cohort studies found a statistically significant protective result for both fruit (RR= 0.74, 95% CI= 0.69-0.81) and vegetable (RR= 0.81, 95% CI= 0.75-0.87) consumption.¹⁶ Fruit (RR= 0.69, 95% CI= 0.62-0.77) and vegetable (RR= 0.78, 95% CI= 0.71-0.86) intake was significantly protective in case-control studies.¹⁶ However, this result was weaker and non-significant in cohort studies (RR for fruit= 0.89, 95% CI= 0.73-1.09; RR for vegetables= 0.89, 95% CI= 0.75-1.05).¹⁶

Colorectal cancer

One meta-analysis revealed a small decreased risk of colorectal cancer, which was significant for higher intakes of fruit and vegetables when all studies were pooled (Table 2).¹⁶ This protective effect was significantly stronger in case-control studies than in cohort studies.¹⁶ In the analyses by cancer site (colon versus rectum), cohort studies found a significant protective effect of vegetables on colon but not rectal cancer.¹⁶ There was insufficient data from case-control studies to calculate the RR for rectal cancer and fruit consumption. Fruit consumption showed stronger protection against rectal cancer than colon cancer in cohort studies.¹⁶

Table 2. Results from meta-analysis on fruit and vegetable intake showing relative risk (RR) and 95% confidence interval (CI) for colorectal cancer, where n = number of studies.¹⁶

Cancer	Study Type	Fruit		Vegetables	
		n	RR (95% CI)	n	RR (95% CI)
Colorectal	All studies	31	0.94 (0.90-0.98)	46	0.91 (0.86-0.97)
	Cohort	16	0.96 (0.90-1.01)	17	0.96 (0.90-1.05)
	Case-control	15	0.93 (0.87-0.99)	29	0.87 (0.80-0.95)
Colon	All studies	19	0.94 (0.89-1.00)	27	0.91 (0.83-1.00)
	Cohort	9	0.97 (0.91-1.04)	11	0.91 (0.86-0.96)
	Case-control	10	0.90 (0.82-0.99)	17	0.90 (0.78-1.03)
Rectal	All studies	-	-	9	0.95 (0.80-1.11)
	Cohort	5	0.88 (0.81-0.96)	5	1.06 (0.90-1.25)
	Case-control	-	-	4	0.75 (0.51-1.08)

In contrast to these results, a randomised controlled trial found that over four years those following a diet low in fat (20% of total calories) and high in fibre (18g fibre per 1000 kcal), fruit and vegetables (3.5 servings per 1000 kcal) did not have a different rate of recurrent colorectal adenomas when compared to those that were instructed to follow their usual diet and were given a brochure containing general healthy eating advice (unadjusted RR= 1.00, 95% CI= 0.90-1.12).¹⁹ While the dietary assessment data did indicate that the intervention and control groups differed substantially in the consumption of fruit and vegetables, the intervention period was relatively short. It is also possible that dietary intervention might only affect the growth of adenomas once they occur.

Most recently, a large cohort study showed that (over a five year follow-up period) for highest versus lowest intake categories, vegetable intake was significantly associated with a reduced risk of colorectal cancer (RR= 0.82, 95% CI= 0.71-0.94), and total fruit and vegetable intake was associated with a non-significant reduced risk of colorectal cancer in men (RR= 0.91, 95% CI= 0.78-1.05).²⁰ In particular, green leafy vegetables were associated with a significantly lower risk of colorectal cancer in men (RR= 0.86, 95% CI= 0.74-0.99).²⁰ However in women, vegetable intake (RR= 1.12, 95% CI= 0.90-1.38) and total fruit and vegetable intake (RR= 1.08, 95% CI= 0.86-1.35) were associated with a non-significant increased risk of colorectal cancer.²⁰ Fruit intake alone was associated with a small non-significant increased risk of colorectal cancer in both men (RR= 1.06, 95% CI= 0.91-1.23) and women (RR= 1.09, 95% CI= 0.88-1.36).²⁰ The difference in results for men and women may be due to reporting errors, as women may be more likely to over report foods perceived as healthy.

Prostate cancer

A meta-analysis which included 10 cohort or nested case-control studies and 11 case-control studies on the association between prostate cancer and tomato, tomato products or lycopene intake showed moderate protection against prostate cancer among those who consumed large amounts of raw tomato (RR= 0.89, 95% CI= 0.80-1.00).¹³ Interestingly, cohort studies showed a greater protective effect (RR= 0.71, 95% CI= 0.57-0.87) than case-control studies (RR= 0.98, 95% CI= 0.86-1.00).¹³

A similar trend was also seen for lycopene intake.¹³ The RR of prostate cancer among those who consumed large amounts of lycopene was 0.89 (95% CI= 0.81-0.98).¹³ Again cohort studies showed a larger protective effect (RR= 0.84, 95% CI= 0.75-0.95) than case-control studies (RR= 0.98, 95% CI= 0.83-1.16).¹³ Cooked tomatoes appeared to provide even greater protection, which could be due to increases in concentration or improved availability of lycopene (RR= 0.81, 95% CI= 0.71-0.92).¹³ In addition, increasing concentrations of serum lycopene showed greater protection against prostate cancer than that of dietary lycopene intake (RR= 0.74, 95% CI= 0.59-0.92).¹³ This effect was even stronger for case-control studies (RR= 0.55, 95% CI= 0.32-0.94).¹³ The influence of dietary lycopene intake on serum lycopene concentrations was not explored in this meta-analysis, however other studies have shown that dietary sources of lycopene can increase serum lycopene levels and reduce oxidative stress effectively.²¹

The association between fruit and vegetable consumption and prostate cancer risk was also investigated as part of the EPIC cohort study.²² The consumption of fruit, vegetables, cruciferous vegetables and combined total fruits and vegetables with prostate cancer risk was examined, however no significant associations were found. The RR in the highest quintile of consumption compared with the lowest quintile was 1.06 (95% CI= 0.84-1.34) for fruits, 1.00 (95% CI= 0.81-1.22) for vegetables, 1.00 (95% CI= 0.79-1.26) for fruits and vegetables combined and 1.01 (95% CI= 0.83-1.23) for cruciferous vegetables.²²

Breast cancer

There was no significant association found between breast cancer risk and vegetable intake, fruit intake or fruit and vegetable juice consumption in the EPIC cohort study.²³ RR estimates (adjusted for breast cancer risk factors) for comparisons of the highest versus the lowest quintiles were 0.98 (95% CI= 0.84-1.14) for total vegetables, 1.09 (95% CI= 0.94-1.25) for total fruit and 1.05 (95% CI= 0.92-1.20) for fruit and vegetable juice.²³

When vegetable intake was divided into specific groupings (leafy, fruiting, root, cabbages, mushrooms and garlic and onions) there was also no evidence for a significant inverse association between intake and breast cancer risk.²³ Adjusted RR estimates ranged from 0.98 (95% CI= 0.85-1.14) for mushrooms to 1.18 (95% CI= 1.01-1.38) for cabbage.²³

A meta-analysis completed in 2003 found that fruit did not provide protection against breast cancer, however this was not the case for vegetables.¹⁶ Increasing fruit consumption by 100g/day (which is approximately one serve per day) had very little association with breast cancer risk (RR= 0.99, 95% CI= 0.98-1.00), whereas increasing vegetable consumption by 100g/day did slightly lower the risk of breast cancer (RR= 0.96, 95% CI= 0.94-0.98).¹⁶ Case-control studies showed a protective effect of vegetables (RR= 0.86, 95% CI= 0.78-0.94), but not cohort studies (RR= 1.00, 95% CI= 0.97-1.02).¹⁶ Fruit consumption was not found to be significantly protective in either the pooled estimate from case-control studies (RR= 0.92, 95% CI= 0.84-1.01) or cohort studies (RR= 0.99, 95% CI= 0.98-1.00).¹⁶

Ovarian cancer

In the only meta-analysis identified, total fruit and vegetable intake was not significantly associated with ovarian cancer risk.²⁴ Twelve cohort studies were included in this analysis. The pooled multivariate RR comparing the highest versus the lowest quartiles of intake showed little association for total fruit (RR= 1.11, 95% CI= 0.89-1.37) and total vegetable (RR= 0.88, 95% CI= 0.71-1.09) consumption with ovarian cancer cases that occurred within the first five years.²⁴

When fruit and vegetable intake were represented as per 100g/day (which is approximately one serve per day), the RR values for total fruits, total vegetables and total fruits and vegetables had no association with ovarian cancer.²⁴ When grouped according to classes of fruit and vegetables, no statistically significant association was found. A marginally significant association with the consumption of green leafy vegetables for a 100g increment was found (pooled multivariate RR= 0.88, 95% CI= 0.76-1.00).²⁴

Lung cancer

The relationship between lung cancer and dietary carotenoids was investigated by analysing the primary data from seven large cohort studies in North America and Europe as part of the Pooling Project of Prospective Studies of Diet and Cancer.²⁵ The intakes of five carotenoids (α -carotene β -carotene, β -cryptoxanthin, lutein and lycopene) were inversely associated with lung cancer risk.²⁵ The associations were similar after adjustment for various variables but were attenuated and no longer significant after additional adjustment for smoking status, duration and quantity. Only the inverse association between β -cryptoxanthin intake and lung cancer risk remained statistically significant (RR= 0.76, 95% CI= 0.67-0.86, $p < 0.001$).²⁵ This result did not change after adjustment for intakes of vitamin C, folate, other carotenoids or multivitamin use, and was also consistent for smoking status. Therefore foods high in β -cryptoxanthin, such as orange juice, oranges and papaw,²⁶ may modestly lower the risk of lung cancer.

The effect of fruit and vegetable consumption and lung cancer risk was also investigated as part of the EPIC study.²⁷ There was a significant inverse association between fruit consumption and lung cancer risk, but no association was found for vegetable consumption after adjustment for age, smoking, height, weight and gender. The HR for lung cancer and fruit consumption was 0.60 (95% CI= 0.46-0.78, p value < 0.01).²⁷ This relationship was strengthened when lung cancers diagnosed in the first two years of follow-up were excluded from the analysis (HR 0.50, 95% CI= 0.36-0.70).²⁷ The HR for lung cancer and vegetable consumption was 1.00 (95% CI= 0.76-1.30).²⁷ There was a non-significant inverse association between leafy vegetables and lung cancer risk (HR= 0.89, 95% CI= 0.66-1.19), and a non-significant positive association between cruciferous vegetables and lung cancer risk (HR= 1.21, 95% CI= 0.92-1.60).²⁷

When data was divided into smoking status (current, ex-smokers, life-long non-smokers), fruit consumption provided a significant inverse association with lung cancer for smokers (HR= 0.51, 95% CI= 0.35-0.73) and life-long non-smokers (HR= 0.33, 95% CI= 0.13-0.83).²⁷ There was a small non-significant positive relationship seen for fruit consumption and lung cancer for ex-smokers (HR= 1.07, 95% CI= 0.65-1.76).²⁷ Vegetable consumption appeared to have a non-significant inverse association with lung cancer for smokers (HR= 0.80, 95% CI= 0.55-1.17), little association for life-long non-smokers (HR= 0.99, 95% CI= 0.45-2.21) and a non-significant positive association for ex-smokers (HR= 1.29, 95% CI= 0.78-2.14).²⁷

The association between fruit and vegetable intake and the risk of lung cancer was investigated as part of the Pooling Project of Prospective Studies of Diet and Cancer.²⁸ Fruit consumption was highest among never smokers and lowest among current smokers. For vegetables, intakes among never and past smokers were generally similar and exceeded intakes among current smokers. Significantly reduced risks of lung cancer were observed among those with higher total fruit (RR= 0.44, 95% CI= 0.38-0.50, *p* trend <0.001), total vegetable (RR= 0.71, 95% CI= 0.63-0.80, *p* trend <0.001) and total fruit and vegetable intakes (RR= 0.48, 95% CI= 0.43-0.54, *p* trend <0.001) on age-adjusted analysis.²⁸ This result was slightly weaker, although still significant when smoking status (never, past, current) was taken into account (RR total fruit= 0.66, 95% CI= 0.58-0.75, *p* trend <0.001; RR total vegetables= 0.81, 95% CI= 0.72-0.92, *p* trend 0.004; RR total fruit and vegetables= 0.67, 95% CI= 0.59-0.77, *p* trend <0.001).²⁸

In 2003, a meta-analysis found that vegetable consumption appeared to provide significant protection against lung cancer (RR= 0.89, 95% CI= 0.82-0.93) with case-control studies (RR= 0.85, 95% CI= 0.77-0.92) showing a stronger protection compared to cohort studies (RR= 0.92, 95% CI= 0.84-1.07).¹⁶ Fruit consumption also provided significant protection against lung cancer (RR= 0.85, 95% CI= 0.78-0.92).¹⁶ This was also reflected in case-controls studies (RR= 0.83, 95% CI= 0.74-0.94) and cohort studies (RR= 0.86, 95% CI= 0.78-0.94).¹⁶ Interestingly, fruit consumption appeared to have a significant protective effect in men that was not found in women. The results for vegetables did not differ by sex.

Bladder cancer

Fruit consumption provided significant protection against bladder cancer (RR= 0.81, 95% CI= 0.73-0.91), which was consistent across five case-control (RR= 0.82, 95% CI= 0.70-0.94) and three cohort studies (RR= 0.80, 95% CI= 0.65-0.99).¹⁶ Vegetable consumption showed a non-significant inverse association across all studies (RR= 0.91, 95% CI= 0.82-1.00), case-control (RR= 0.90, 95% CI= 0.78-1.03) and cohort studies (RR= 0.92, 95% CI= 0.75-1.14).¹⁶

Potential mechanisms of action

Many possible mechanisms have been proposed to account for protection by fruit and vegetables, particularly as the terms “fruit” and “vegetable” include a broad range of foods, and fruit and vegetables contain a large range of biologically active compounds. Protective dietary components include fibre, vitamins, minerals, antioxidants and phytochemicals like carotenoids, flavonoids, isoflavonoids, allium compounds and dithiolthiones.

Nutrients found in fruit and vegetables may help to lower the risk of cancer by:²⁹⁻³³

- Helping to reduce the oxidative damage to DNA caused by free radicals;
- Interacting with carcinogens e.g. reducing the formation and activation, as well as assisting with the detoxification of carcinogens; and
- Altering the activity of various metabolising enzymes and affecting cellular mechanisms important in cancer development.

A comprehensive list of the possible anti-carcinogenic mechanisms of nutrients found in fruit and vegetables can be seen in Table 3.

Table 3. Possible anti-carcinogenic mechanisms of nutrients in fruits and vegetables.²⁹⁻³³

Phytochemical	Proposed mechanism	Food Source
Vitamins C and E, carotenoids, polyphenols	Antioxidant protection against oxidative damage to DNA, cellular macromolecules and membranes.	Fruit and vegetables generally: especially yellow and orange; citrus fruit; berries.
Dithiolthiones, isothiocyanates, allium compounds	Increases Type II detoxifying enzymes (e.g. glutathione S. transferase).	Cruciferous vegetables: broccoli, cauliflower cabbage, brussel sprouts, kohlrabi; allium vegetables: onions, leeks, chives.
Vitamin C, allium compounds	Reduces bacterial formation of nitrosamines from nitrate in stomach. Decreases Type 1 activating enzymes (e.g. aryl hydroxylase).	All fruit and vegetables (Vitamin C) ³⁴ : especially blackcurrants, guava, citrus, kiwi fruit, broccoli, sprouts; allium vegetables: onions, leeks, chives.
Folic acid	Preserve integrity of DNA and ensure optimum DNA methylation.	Green leafy vegetables; avocado; oranges.
Carotenoids, flavonoids	Induction of cell differentiation.	Yellow/orange fruits and vegetables: carrots, sweet potato, mango, pumpkin, red capsicum, rockmelon, paw paw, tomato; dark green vegetables: silverbeet, spinach, broccoli, dark green lettuce, Chinese greens (e.g. bok choy), kale, parsley, basil.
Soluble fibre, resistant starch	Decreases concentrations of secondary bile acids, which modify the enzyme activities of intestinal bacteria. Fermentation, which produces short-chain fatty acids that may inhibit carcinogenesis via effects on colonic pH and increased availability of butyrate.	All fruits and vegetables (including legumes), particularly: Soluble fibre ³⁵ : dried apricots, dried figs, prunes, quince, okra, cabbage, carrot, broccoli, leeks, brussel sprouts, beetroot, lychees, peas, mulberries, asparagus, lemons, oranges, swede, parsnip, dates, plums. Resistant starch ³⁶ : corn, bananas, peas, potato, sweet potato, broad beans.
Insoluble fibre	Dilutes carcinogens by increasing faecal bulking. Reduces interaction of carcinogens with mucosal cells by increasing stool transit time.	All fruits and vegetables, particularly ³⁵ : guava, quince, peas, dried figs, corn, broad beans, berries, dates, pears, prunes, cabbage, spinach, pineapple, broccoli, onion, leek, asparagus, turnip, swede, beetroot, squash, brussel sprouts, okra, carrots, pumpkin, rhubarb, green beans.

Recommendations

Cancer Council supports the Australian Dietary Guidelines that recommend eating plenty of fruit and vegetables. Because knowledge is still incomplete about the ways in which phytochemicals may reduce cancer risk and their potential relevance to specific tissues and to particular stages of cancer development, Cancer Council also recommends that people eat a **variety** of different fruit and vegetables to obtain maximum benefits.

In defining the variety of fruits and vegetables to recommend, attention should be paid to their phytochemical content as well as to the epidemiological evidence concerning their protective potential. On this basis fruits should include citrus fruits, coloured fruits (especially red, yellow and orange) and berries, while vegetables should include cruciferous and allium types, dark-green leafy vegetables and red/yellow/orange vegetable types. The value of eating raw and cooked vegetables instead of taking dietary supplements has been repeatedly shown.³⁷ Therefore both cooked and raw vegetables should be recommended in a cancer protective diet.

The definitions for fruit in many studies include fruit juices. The cancer protective phytochemicals are preserved in fresh fruit and vegetable juices^{38, 39} and some phytochemicals may be more bioavailable in juices rather than whole fruits and vegetables.⁴⁰ However the fibre is removed in most vegetable and fruit juices and also in dietary supplements, and fibre is thought to be protective against colon cancer.^{41, 42} Therefore fruit and vegetables are best consumed whole, rather than as a juice or individual nutrients in a supplement form. As well, studies suggest that antioxidant supplements are not protective and may in fact increase overall mortality.⁴³

How much fruit and vegetables should we be eating?

The population recommendation of at least **two serves of fruit** and **five serves of vegetables** daily^{44, 45} is appropriate for cancer prevention. For many Australians, this means doubling their current intake. Table 4 shows examples of a serve of fruit and vegetables, as specified in The Australian Guide to Healthy Eating.⁴⁵

Table 4. Sample fruit and vegetable serving sizes in The Australian Guide to Healthy Eating.⁴⁵

Fruit	1 serve equals: <ul style="list-style-type: none"> • 1 medium piece (150g) of fruit (apple, banana, orange, pear) • 2 small pieces (150g) of fruit (apricots, kiwifruit, plums) • 1 cup (150g) diced pieces or canned fruit • 1½ tablespoons sultanas, 4 dried apricot halves • ½ cup (125mL) fruit juice
Vegetables	1 serve equals: <ul style="list-style-type: none"> • ½ cup (75g) cooked vegetables • ½ cup (75g) cooked dried beans, peas or lentils • 1 cup salad vegetable • 1 small potato

As stated in the 2007 WCRF/AICR review, there is no *convincing* evidence that higher intakes of fruit and vegetables are harmful, and almost all findings are to the contrary.⁴ Protection is often, but not always, found in the upper quartile or quintile of consumption, which in many studies equates approximately to the recommended 2-3 servings of fruit and 4-5 servings of vegetables.

Because some of the proposed mechanisms for how fruit and vegetables protect against cancer work at the early stages of cancer development and initiation, and most cancers develop over many years, it is important that a protective diet begins in childhood and extends into adulthood.

In addition to their anti-carcinogenic activity, fruit and vegetables play an important role in weight regulation due to their low energy density and fibre content. As obesity emerges as a major risk factor for several cancers, the importance of fruit and vegetables will continue to strengthen.

Current Australian consumption levels

The 1995 National Nutrition Survey indicated that on average, only 28% of Australians consumed at least 300g (two serves) of fruit on the day of the survey, and this included fruit juices, while only 32% ate at least 300g (four serves) of vegetables, even when potatoes were included.⁴⁶ The survey also found that nearly half of all Australian adults ate no fruit and one in ten ate no vegetables on any given day.⁴⁶

The 2006 NSW Health Survey showed that across NSW the number of people aged 16 years and over eating the recommended two serves of fruit each day had increased from 46.1% in 1997 to 53.4% in 2006, and the number eating three or more serves of vegetables each day had increased from 34.0% in 1997 to 40.9% in 2006.⁴⁷

The barriers to consuming fruit and vegetables include high prices (especially of fruit), time taken to prepare vegetables, inadequate supply and quality, concerns about pesticides, and social and domestic household changes which result in less time being available to prepare food.⁴⁸ Concerns about pesticides are almost certainly unwarranted as Australian produce consistently shows low levels of chemical residues and contaminants, which are well below acceptable safety limits.⁴⁹

Cancer survivors

There are a small number of studies indicating that diet may play a role in preventing cancer progression and recurrence in people who already have cancer, however intervention studies with cancer end-points are few in number and further research is needed before definitive dietary advice can be given to cancer patients.⁵⁰

However the Women's Healthy Eating and Living (WHEL) study, a randomised controlled trial evaluating the effect of an increased intake of fruit, vegetables and fibre among both pre- and postmenopausal women with early-stage breast cancer, did not find a statistically significant protective association between those who increased their fruit and vegetable consumption and those who did not.⁵¹ After an average of follow up period of 7.3 years, there were no differences in the risk of recurrence of breast cancer ($p=0.63$) or in the risk of overall mortality ($p=0.43$) between the two groups. The women in both groups of the WHEL study experienced small weight gains, so it may be possible that preventing further weight gain may have more of an impact on breast cancer recurrence and survival than just boosting fruit and vegetable intake.

The American Cancer Society have issued advice to cancer survivors, particularly of breast, lung, and colorectal cancers, to adopt the general cancer preventative recommendations for fruit and vegetables consumption, although they acknowledge there are few studies that have examined whether this improves cancer survival.⁵² WCRF also recommends cancer survivors follow the recommendations for diet and cancer prevention.⁴

Organic fruit and vegetables

Organic foods are produced without using synthetic chemicals such as pesticides, fertilisers, hormones and antibiotics or genetic modification. There is also an emphasis on appropriate land management and use of renewable resources and conservation practices. Reasons why people choose organic fruits and vegetables include reduced environmental impact, avoidance of genetic modification, better flavour and taste, perceived health benefits or just personal preference. However organic foods are more expensive than conventional foods, and there is also potentially higher wastage due to a shorter shelf life of some products.

It is difficult to measure accurately the nutritional content of organic versus conventionally grown food due to the variables that affect the end product. Such variables include types of fertilisers used, soil types, harvesting methods, transport and storage. However it appears that organic produce may provide higher levels of vitamin C and lower nitrate levels compared with conventionally produced fruits and vegetables.⁵³

Presently there is no evidence to suggest that organic fruit and vegetables are more effective in reducing cancer risk than conventionally grown fruit and vegetables. Therefore the consumption of all fruits and vegetables should be encouraged, whether they are organic or not. The consumption of organic fruit and vegetables are an individual's choice. It is recommended that all fruits and vegetables be washed and peeled where appropriate to remove possible pesticide residue and possible microbial growth.

Future research

More studies are needed on the association between fruit, vegetables and cancer risk. In the future, there is a need for more studies that:

- Are prospective and include a very large follow-up period;
- Examine the reasons for differences seen between cohort and case-control study results;
- For certain cancers, investigate further the difference in risk for men and women;
- Explore the risk associated with sub-groups of fruit and vegetables e.g. citrus fruit, green leafy vegetables, cruciferous vegetables;
- Consider broad dietary patterns e.g. those that eat small amounts of fruit and vegetables may also eat small amounts of dietary fibre;
- Investigate ways in which individual phytochemicals may reduce cancer risk;
- Examine whether fruit and vegetables play a role in preventing cancer progression and recurrence in people who already have cancer; and
- Identify intermediate biomarkers that are suitable for inferring cancer outcomes or use the molecular profile of certain cancers (e.g. colorectal) as endpoints in order to help clarify inconsistent results in the literature.

Further information

Cancer Council New South Wales
PO Box 572 Kings Cross NSW 1340
www.cancercouncil.com.au
ABN 51 116 463 846

Contact

Kathy Chapman, Nutrition Program Manager: kathyc@nswcc.org.au
Hayley Griffin, Nutrition Project Officer: hayleyg@nswcc.org.au

Acknowledgements

This position statement has been reviewed by:

- Bruce Armstrong
- Finlay Macrae
- Vicki Flood
- Simone Lee
- Freddy Sitas
- Carla Saunders

Cancer Council Australia, GPO Box 4708, Sydney NSW 2001
Ph: (02) 8063 4100 Fax: (02) 8063 4101 Website: www.cancer.org.au

References

1. Strategic Inter-Governmental Nutrition Alliance. Eat Well Australia: a strategic framework for public health nutrition. Canberra, National Public Health Partnership. 2001.
2. National Health and Medical Research Council. Dietary Guidelines for Australian Adults. National Health and Medical Research Council. Canberra. 2003.
3. International Agency for Research on Cancer. *Weight control and physical activity*. Volume 6. Lyon: IARC. 2002.
4. World Cancer Research Fund and American Institute for Cancer Research. Food, nutrition, physical activity and the prevention of cancer: a global perspective. Washington DC, AICR. 2007.
5. World Health Organization. Diet, nutrition and the prevention of chronic diseases. Geneva, World Health Organisation. 2003.
6. International Agency for Research on Cancer. *Fruit and Vegetables*. Volume 8. Lyon: IARC. 2003.
7. Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez A. The Burden of Disease and Injury in Australia 2003. Canberra, Australia, Australian Institute of Health and Welfare (AIHW). 2007.
8. Marks G, Pang G, Coyne T, Picton P. Cancer Costs in Australia - the potential impact of dietary change. Canberra, Australian Food and Nutrition Monitoring Unit, Commonwealth Department of Health and Aged Care. 2001.
9. United Kingdom Department of Health. Nutritional aspects of the development of cancer. Report of the working group on diet and cancer of the Committee on Medical Aspects of the Food and Nutrition Policy. Committee on Medical Aspects of the Food and Nutrition Policy. Norwich, UK, The Stationery Office. 1998.
10. World Cancer Research Fund and American Institute for Cancer Research. *Food, nutrition and the prevention of cancer: a global perspective*. New York: American Institute for Cancer. 1997.
11. International Agency for Research on Cancer. *Cruciferous Vegetables, Isothiocyanates and Indoles*. Volume 9. Lyon: IARC. 2004.
12. Khaw KT, Bingham S, Welch A, Luben R, Wareham N, Oakes S *et al*. Relation between plasma ascorbic acid and mortality in men and women in EPIC-Norfolk prospective study: a prospective population study. European Prospective Investigation into Cancer and Nutrition. *Lancet*. 2001; 357(9257): 657-663.
13. Etminan M, Takkouche B, Caamano-Isorna F. The role of tomato products and lycopene in the prevention of prostate cancer: a meta-analysis of observational studies. *Cancer Epidemiol Biomarkers Prev*. 2004; 13(3): 340-345.
14. Hung HC, Joshipura KJ, Jiang R, Hu FB, Hunter D, Smith-Warner SA *et al*. Fruit and vegetable intake and risk of major chronic disease. *J Natl Cancer Inst*. 2004; 96(21): 1577-1584.
15. Pavia M, Pileggi C, Nobile CG, Angelillo IF. Association between fruit and vegetable consumption and oral cancer: a meta-analysis of observational studies. *Am J Clin Nutr*. 2006; 83(5): 1126-1134.
16. Riboli E, Norat T. Epidemiologic evidence of the protective effect of fruit and vegetables on cancer risk. *Am J Clin Nutr*. 2003; 78(3 Suppl): 559S-569S.
17. Gonzalez CA, Pera G, Agudo A, Bueno-de-Mesquita HB, Ceroti M, Boeing H *et al*. Fruit and vegetable intake and the risk of stomach and oesophagus adenocarcinoma in the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST). *Int J Cancer*. 2006; 118(10): 2559-2566.
18. Lunet N, Lacerda-Vieira A, Barros H. Fruit and vegetables consumption and gastric cancer: a systematic review and meta-analysis of cohort studies. *Nutr Cancer*. 2005; 53(1): 1-10.
19. Schatzkin A, Lanza E, Corle D, Lance P. Lack of effect of a low-fat, high-fiber diet on the recurrence of colorectal adenomas. *NEJM*. 2000; 342(16): 1149-1156.
20. Park Y, Subar AF, Kipnis V, Thompson FE, Mouw T, Hollenbeck A *et al*. Fruit and Vegetable Intakes and Risk of Colorectal Cancer in the NIH-AARP Diet and Health Study. *Am J Epidemiol*. 2007; 166(2): 170-180.
21. Rao AV. Processed tomato products as a source of dietary lycopene: bioavailability and antioxidant properties. *Can J Diet Pract Res*. 2004; 65(4): 161-165.
22. Key TJ, Allen N, Appleby P, Overvad K, Tjonneland A, Miller A *et al*. Fruits and vegetables and prostate cancer: no association among 1104 cases in a prospective study of 130544 men in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Int J Cancer*. 2004; 109(1): 119-124.
23. van Gils CH, Peeters PH, Bueno-de-Mesquita HB, Boshuizen HC, Lahmann PH, Clavel-Chapelon F *et al*. Consumption of vegetables and fruits and risk of breast cancer. *JAMA*. 2005; 293(2): 183-193.
24. Koushik A, Hunter DJ, Spiegelman D, Anderson KE, Arslan AA, Beeson WL *et al*. Fruits and vegetables and ovarian cancer risk in a pooled analysis of 12 cohort studies. *Cancer Epidemiol Biomarkers Prev*. 2005; 14(9): 2160-2167.
25. Mannisto S, Smith-Warner SA, Spiegelman D, Albanes D, Anderson K, van den Brandt PA *et al*. Dietary carotenoids and risk of lung cancer in a pooled analysis of seven cohort studies. *Cancer Epidemiol Biomarkers Prev*. 2004; 13(1): 40-48.

26. Manzi F, Flood V, Webb K, Mitchell P. The intake of carotenoids in an older Australian population: The Blue Mountains Eye Study. *Public Health Nutr.* 2002; 5(2): 347-352.
27. Miller AB, Altenburg HP, Bueno-de-Mesquita B, Boshuizen HC, Agudo A, Berrino F *et al.* Fruits and vegetables and lung cancer: Findings from the European Prospective Investigation into Cancer and Nutrition. *Int J Cancer.* 2004; 108(2): 269-276.
28. Smith-Warner SA, Spiegelman D, Yaun SS, Albanes D, Beeson WL, van den Brandt PA *et al.* Fruits, vegetables and lung cancer: a pooled analysis of cohort studies. *Int J Cancer.* 2003; 107(6): 1001-1011.
29. Greenwald P, Clifford CK, Milner JA. Diet and cancer prevention. *European Journal of Cancer.* 2001; 37(8): 948-965.
30. Heber D. Vegetables, fruits and phytoestrogens in the prevention of diseases. *J Postgrad Med.* 2004; 50(2): 145-149.
31. Johnson IT. Micronutrients and cancer. *Proc Nutr Soc.* 2004; 63(4): 587-595.
32. Smith TA. Carotenoids and cancer: prevention and potential therapy. *Br J Biomed Sci.* 1998; 55(4): 268-275.
33. Talalay P, Fahey JW. Phytochemicals from cruciferous plants protect against cancer by modulating carcinogen metabolism. *J Nutr.* 2001; 131(11 Suppl): 3027S-3033S.
34. National Health and Medical Research Council. Nutrient Reference Values for Australia and New Zealand including Recommended Dietary Intakes. Canberra, Australia, Commonwealth Department of Health and Ageing. 2006.
35. Englyst HN, Cummings JH. Improved method for measurement of dietary fiber as non-starch polysaccharides in plant foods. *J Assoc Off Anal Chem.* 1988; 71(4): 808-814.
36. Record S. CSIRO Australia. Personal communication on resistant starch. 2002.
37. Link LB, Potter JD. Raw versus cooked vegetables and cancer risk. *Cancer Epidemiol Biomarkers Prev.* 2004; 13(9): 1422-1435.
38. Helsler MA, Hotchkiss JH, Roe DA. Influence of fruit and vegetable juices on the endogenous formation of N-nitrosoproline and N-nitrosothiazolidine-4-carboxylic acid in humans on controlled diets. *Carcinogenesis.* 1992; 13(12): 2277-2280.
39. Xu GP, Song PJ, Reed PI. Effects of fruit juices, processed vegetable juice, orange peel and green tea on endogenous formation of N-nitrosoproline in subjects from a high-risk area for gastric cancer in Moping County, China. *Eur J Cancer Prev.* 1993; 2(4): 327-335.
40. McEligot AJ, Rock CL, Shanks TG, Flatt SW, Newman V, Faerber S *et al.* Comparison of serum carotenoid responses between women consuming vegetable juice and women consuming raw or cooked vegetables. *Cancer Epidemiol Biomarkers Prev.* 1999; 8(3): 227-231.
41. Steinmetz KA, Potter JD. Vegetables, fruit, and cancer prevention: a review. *J Am Diet Assoc.* 1996; 96(10): 1027-1039.
42. Bingham SA, Day NE, Luben R, Ferrari P, Slimani N, Norat T *et al.* Dietary fibre in food and protection against colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC): an observational study. *Lancet.* 2003; 361(9368): 1496-1501.
43. Bjelakovic G, Nikolova D, Gluud LL, Simonetti RG, Gluud C. Mortality in randomized trials of antioxidant supplements for primary and secondary prevention: systematic review and meta-analysis. *JAMA.* 2007; 297(8): 842-857.
44. National Health and Medical Research Council. Dietary Guidelines for Australian Adults. National Health and Medical Research Council. Canberra. 2003.
45. Children's Health Development Foundation & Deakin University. The Australian guide to healthy eating. Canberra, AGPS. 1998.
46. McLennan W, Podger A. National Nutrition Survey: Foods Eaten, Australia 1995. Canberra, Australian Bureau of Statistics. 1999.
47. Centre for Epidemiology and Research. NSW Population Health Survey: 2006 Summary Report on Adult Health by Area Health Service. Sydney, NSW Department of Health. 2007.
48. Cox DN, Beaumont-Smith N, Baghurst K. An issues paper on barriers to the consumption of vegetables and fruits. Canberra, Commonwealth Department of Health and Aged Care. 1999.
49. Food Standards Australia New Zealand (FSANZ). The 20th Australian Total Diet Survey. Canberra, FSANZ. 2002.
50. Davies AA, Davey Smith G, Harbord R, Bekkering GE, Sterne JAC, Beynon R *et al.* Nutritional interventions and outcome in patients with cancer or preinvasive lesions: Systematic review. *Journal of the National Cancer Institute.* 2006; 98(14): 961-973.
51. Pierce JP, Natarajan L, Caan BJ, Parker BA, Greenberg ER, Flatt SW *et al.* Influence of a diet very high in vegetables, fruit, and fiber and low in fat on prognosis following treatment for breast cancer: the Women's Healthy Eating and Living (WHEL) randomized trial. *JAMA.* 2007; 298(3): 289-298.

52. Doyle C, Kushi LH, Byers T, Courneya KS, Demark-Wahnefried W, Grant B *et al.* Nutrition and physical activity during and after cancer treatment: an american cancer society guide for informed choices. *CA Cancer J Clin.* 2006; 56(6): 323-353.
53. Williams CM. Nutritional quality of organic food: shades of grey or shades of green? *Proc Nutr Soc.* 2002; 61(1): 19-24.